

**Amendments to the Specification:**

Please replace the text of Table 2 on page 31, in the box at column 4, row 4 (including the Title row) with the following amended text:

US Patent Appl. No. 09/123,251 (pending)

Please replace the text of Table 3 on page 33, in the box at column 4, row 7 (including the Title row) with the following amended text:

US Patent Appl. No. 09/123,251 (pending)

Please replace the paragraph beginning at page 4, line 26 with the following amended paragraph:

The invention also provides a recombinant microorganism expressing one or more exogenous sugar transferases, or one or more exogenous nucleotide sugar precursor ~~synthesising~~ synthesizing enzymes, said microorganism also expressing an acceptor molecule, said one or more exogenous sugar transferases being specific for the transfer of one or more sugar residues represented progressively from a non reducing terminal end of a receptor of either a toxin or an adhesin of a pathogenic organism, the exogenous sugar transferases progressively transferring said one or more sugar residues onto the acceptor molecule to thereby form a chimeric carbohydrate molecule with an exposed receptor mimic, said sugar precursor enzymes forming nucleotide precursors that are transferred to said acceptor molecule to make up said chimeric carbohydrate, said exposed receptor mimic capable of binding the toxin or the adhesin.

Please replace the paragraph beginning at page 59, line 25 with the following amended paragraph:

*C. difficile* infection is associated with broad spectrum antibiotic therapy and is the commonest cause of infectious diarrhoea and life-threatening pseudomembranous colitis in hospitalized patients. Antibiotic therapy permits overgrowth of the gut by this bacterium, which elaborates two potent cytotoxins (exotoxins A and B). Exotoxin A is enterotoxic and is essential for human virulence; exotoxin B can only damage host tissues after destruction of the epithelial barrier by exotoxin A (53). *C. difficile* exotoxin A binds to several human glycolipids, all of which contain a  $\text{Gal}\beta[1\rightarrow4]\text{GlcNAc}$   ~~$\text{Gal}\beta[1\rightarrow4]\text{GlcNAc}$~~  moiety. Genes encoding transferases capable of assembling this epitope are also found in the *Neisseria lgt* locus. Expression of *lgtABE* in *E. coli*

CWG308 was predicted to result in synthesis of a LPS outer core oligosaccharide comprising Gal $\beta$ [1 $\rightarrow$ 4]GlcNAc $\beta$ [1 $\rightarrow$ 3] Gal $\beta$ [1 $\rightarrow$ 4]Glc $\rightarrow$  (lacto-*N*-neotetraose). The *lgtA-B* genes were amplified from *N. gonorrhoeae* DNA by PCR, and the poly-G tract in *lgtA* was mutagenized by overlap-extension PCR, as follows. The 5' portion of *lgtA* was amplified using primers 5'-CAGGCGAATTCAAATTATCGGGAGAGTA-3' (LGTAFF) (SEQ ID NO:21) incorporating an *Eco*RI site (underlined) and 5'-ATATTCGCCACCGCCACCGCCCGACTTTGCCAATTCG-3' (LGTAOLR) (SEQ ID NO:22). The 3' portion of *lgtA* and all of *lgtB* was amplified using primers 5'-GTCGGGCGGTGGCGGT GGCGAATATATTGCGCGCACCG-3' (LGTAOLF) (SEQ ID NO:23) and 5'-CATCTTGGATCC TTTTATTGGAAAGGCAC-3' (LGTBR) (SEQ ID NO:24) incorporating a *Bam*HI site (underlined). PCR was performed using the Expand<sup>TM</sup> High Fidelity PCR System (Roche Molecular Biochemicals) under conditions recommended by the supplier. The two PCR products were then purified, aliquots were mixed, and the complete *lgtAB* coding sequence with the desired modifications was amplified using primers LGTAFF and LGTBR. This procedure mutates the four consecutive GGG codons in the poly-G tract in *lgtA* to GGT or GGC (all of which encode Gly), eliminating the risk of slipped-strand mispairing without changing the encoded amino acid sequence. That part of the *lgtA* gene sequence that has been altered is shown below, the sequence shown is taken from nucleotide 699 to 715 of GenBank accession number U14554, altered nucleotides are shown in bold and underlined. GGGCGGTGGCGGTGGCG (SEQ ID NO:25). The modified PCR product was then digested with *Eco*RI and *Bam*HI and cloned into the similarly digested derivative of pK184 containing *lgtE* described earlier. This places *lgtAB* between the pK184 vector promoter and *lgtE*, with all three genes in the same orientation such that they will be co-transcribed. Insertion of *lgtAB* with the correct mutations and the appropriate orientation was confirmed by sequence analysis of plasmid DNA using custom made oligonucleotide primers and dye-terminator chemistry on an ABI model 377 automated DNA sequencer. This plasmid (designated pJCP-LNT) was then transformed into *E. coli* CWG308.